SHORT COMMUNICATION

Electrocardiographic Study of 20 Champion Swimmers Before and After 110-Yard Sprint Swimming Competition

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ABSTRACT

In order to record the strain of actual competition, 20 healthy young athletes were studied by serial electrocardiograms recorded after swimming races of 110 yards. Blood pressures and electrocardiograms were taken at 3, 6, 9, 12, 15, 20, 25 and 30 minutes after each race. Of the 20 cases studied, 18 showed at least one of the following abnormal findings: (1) The PR interval was prolonged by at least 0.04 second and up to 0.10 second (12 subjects). (2) "True" ST depression (two subjects) or "false" ST depression (six subjects) was seen. (3) A marked loss of T potential (all subjects) or T inversion (eight subjects), which in some had developed as late as the 12-minute reading, was recorded. The causes for these alterations are considered physiological under the conditions of this experiment.

ALTHOUGH the electrocardiogram has been used to study the recovery of the athlete's heart after strenuous exertion,[1-4] most studies have been conducted under laboratory conditions[1-3] and are not therefore indicative of the true strain of athletic competition. While some investigations[5-8] have measured the stress of actual competition, the graph readings during these reports either were inconsistently spaced[5, 6] or were not taken continuously,[7, 8] being limited to but one recovery reading. This communication reports the findings on athletes under competitive conditions.

METHOD

Nineteen highly trained and healthy teen-age swimmers of championship calibre were selected for study before and after 110-yard sprint swimming competition. One swimmer was studied a second time, two years after the first testing, making a total of 20 sets of observations.

The subjects were studied several days before the exercise to determine their supine, resting blood pressures and pulse rate and to record their supine, resting electrocardiograms. The swimming races were, for the most part, held in regularly scheduled meets and were distances of 110 yards. Four observations were made following Pan American Games' time trials of 110-yard swims. The study was concerned with the changes in and the recovery aspects of the participants' electrocardiograms, pulse rates, and blood pressures following the race.

The 30-minute observation period began two minutes after the race. Each subject had supine electrocardiograms taken with a Sanborn Viso Cardiette at three, six, nine 12, 15, 20, 25 and 30 minutes following the "all-out" sprint. Supine blood pressure readings were taken at two, five, eight, 11, 17, 19, 22, 27, 29 and 32 minutes.

RESULTS

Pulse rate.—The pulse rate had an average increase of 92.7% above the resting rate. The range of rate increase was from 37% to 225%. Only one of the athletes tested returned to his determined resting rate within the 30-minute recovery period.

The pulse rate of one male subject showed a large increase from 40 to 130 by three minutes. Two other male subjects displayed sharp increases from 55 to 120 and from 75 to 140 beats per minute. The largest increase in a female subject was from 54 to 115. The average pulse rate increase for all subjects was from 60.7 at rest to 112.6 beats per minute at the three-minute reading.

Blood pressure.—The average resting systolic pressure was 117 mm. Hg, which had risen at two minutes after the race to an average of 165 mm. Hg. The average resting diastolic reading was 70 mm. Hg, which had fallen to an average of 47 mm. Hg at the two-minute reading. Three subjects showed an increase in diastolic pressure up to 20 points.

The largest increase was recorded in a 15-year-old boy with a resting reading of 120/60-50. Two minutes after the exercise his pressure was 200/80-0. This subject's pulse rate had risen from a resting rate of 50 to 96 beats per minute at the three-minute interval.

The least of the blood pressure changes was displayed by a 22-year-old man. From a resting position of 130/70 he registered a two-minute reading of 160/40.

Ten of the subjects showed a return to their resting levels by 32 minutes, the others showing a steady trend toward pre-exercise levels.

ELECTROCARDIOGRAM

Rhythm.—Eight of the 20 subjects tested showed a marked sinus arrhythmia before and after exer-

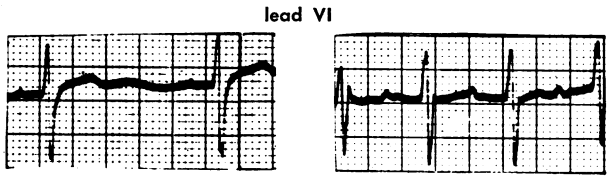

Fig. 1.—Male E.C., PR prolongation.

tion. Seventeen subjects developed marked sinus tachycardia after their race.

P wave.—Five subjects showed no change in their P waves, while 13 showed significant rises in amplitude. Two subjects displayed a 2 mv. increase at three minutes. The two remaining subjects showed P wave loss up to 1 mv.

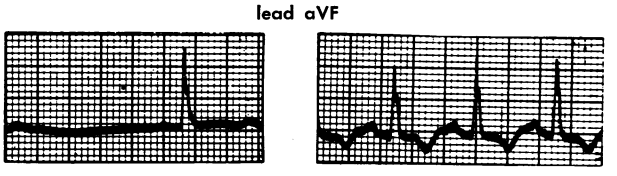

Fig. 2.—Male B.W., T inversion.

PR interval.—Eight subjects showed no change in their PR intervals despite rapid rate increases. In 12 cases there was a prolongation of 0.04 second or more. Two of these had not returned to their resting levels by the 30-minute reading. All subjects were recorded as having values within normal limits prior to competition.

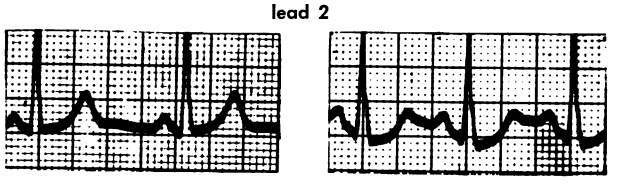

Fig. 3.—Male D.J., "false" ST depression.

A 16-year-old male subject showed an increase of 0.10 second, the PR increasing from 0.14 second at rest to 0.24 second by three minutes.

QRS interval.—The ventricular conduction time seemed little affected in most subjects. Fifteen records showed no changes from the normal interval length. In three cases the duration was increased slightly, while two subjects showed a decrease in time.

ST segment.—Eight subjects appeared to have significantly depressed ST segments up to —0.2 mv. The depression was maximal in the early tracings but persisted for some time in a few subjects. Six of the subjects showing this depression had recovered their resting levels at various times over the 30-minute recovery period. Two subjects did not fully recover, within the period of observation.

However, after applying the criteria proposed by Lepeschkin and Surawicz,[9] it was seen that only two of these subjects showed a "true" ST depression. The alterations shown by the remaining six swimmers would be described as typical "false" ST depressions because of the sloping PR segments.

Six other subjects showed ST segments that were up to 0.15 mv. above isoelectric at rest. For these subjects this segment became isoelectric at three minutes. This segment had returned to the positive resting level by the 30-minute reading in all subjects.

T wave.—The T waves in all cases showed a marked loss of potential. In nine subjects (three females and six males) certain leads had actually become inverted. The inversion shown ranged between —.05 and —.25 mv., averaging —.15 mv. A male Olympic swimmer showed a resting T in lead aVF of 0.05 mv. This lead had inverted to —0.2 mv. at the 12-minute reading. By the final reading it was —0.05 mv.

A total of 13 athletes showed graphs that had not returned to normal in 30 minutes. Only one subject showing inversion had fully recovered by the final reading. In the graphs of one 20-year-old male the T waves were small at 30 minutes and had not returned to normal in 24 hours. One 17-year-old female had returned to resting levels by 24 hours.

Five subjects showed high peaked T waves at three minutes. These diminished quickly and steadily to a position below the amplitude shown in the resting reading.

R and S waves.—Only one female subject showed significant variation in the R and S waves, namely in the height of R and in the depth of S.

U wave.—All subjects displayed a U wave at rest. Only two male subjects showed prominent U waves in the recovery graphs, although finer measuring criteria, as recommended by Holzmann,[10] were not used.

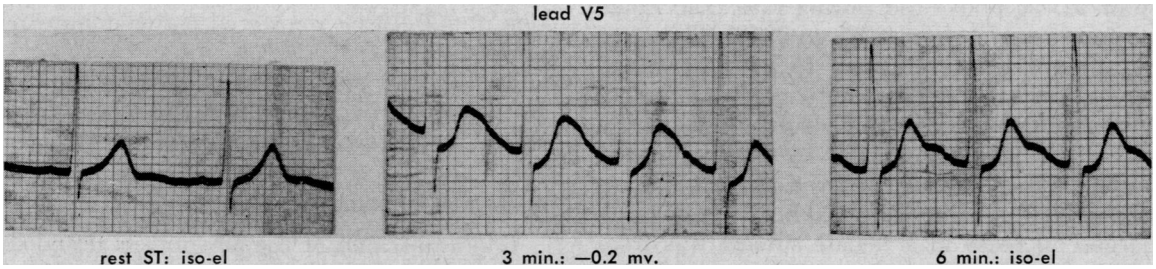

Fig. 4.—Male W.M., "true" ST depression.

ALL WAVES CONSIDERED

Eighteen out of the 20 performances studied produced at least one of the following variations: PR prolongation, T inversion or "true" ST depression. Two male subjects showed insignificant changes in these areas.

DISCUSSION

The first significant finding was in the atrioventricular conduction time. The PR interval was prolonged in 12 of the 20 cases up to 0.24 second. This finding is in contrast to previous reports.[2, 6, 11, 12] The prolongation of this interval is doubly significant because of the increased heart rate that these subjects displayed. Increased rate is ordinarily accompanied by shortened atrioventricular conduction time.

The second significant finding was in the change of the recovery phases of ventricular activity, namely the degree of ST depression, the loss of T wave amplitude and even the occurrence of T wave inversion. Generally speaking, these findings have not been reported in studies of healthy, normal young people following exercise.[3, 5, 8, 13] In the present study it was seen that all of the 20 subjects tested showed a decrease in the T wave amplitude following swimming competition. Furthermore, nine of these healthy young athletes have shown T wave inversion. Also, six subjects showed a "false" ST depression and two a "true" ST depression. Previous observers[3, 5, 12] consider such depression to be an uncommon response.

The 19 individuals involved in this study were free of clinically detectable heart disease. They were all in their late teens, were all in excellent general condition, and were all well trained in their events. Yet they showed prolongation of their AV conduction time and marked ST segment and T wave changes. In this setting, the variations can be interpreted only as manifestations of functional alterations in the heart following such maximal competitive exertion. There was no suggestion that these functional changes were harmful, although recovery in many cases was delayed beyond 30 minutes.

There are differences of opinion as to the gravity of the changes that have been demonstrated by the 20 performances reviewed here.[6, 13-15] Not all would attach serious meaning to T wave inversion and ST depression. Gardberg and Rosen,[14] for example, in 1958 stated that the prejudice held against T wave inversion was traditional and that this anomaly was not necessarily caused by disease. Lozada and Tempone[3] however, have stated, after studying 20 normal men and women who had run in place for one minute, that a depression, appearing as late as five minutes after exercise, had pathological significance. Morin *et al.*[8] in 1951 described T wave and ST segment depression in rugby players which they considered had passed beyond the limits of physiological adaptation.

The excitement and anxiety of competition may have been a causative factor in the production of some of these changes. Also, the breath-holding of speed swimming may be of some importance, since oxygen lack and carbon-dioxide retention are increased thereby to a greater degree than in other competitive sports.

The oxygen debt that develops within the musculature during severe exertion is accompanied by increased lactic acid concentration and carbon-dioxide accumulation which tend to cause acidosis; and a resulting change in the blood pH may affect the cardiogram. It should also be noted that during such sudden, severe muscle strain there is almost always an important shift of electrolytes (particularly potassium) out of the muscle cell.

In 1951 Yu[16] produced ST depression by induced hypoxemia and graded tread-mill walking. Beckner and Winsor[6] studied 10 marathon runners in 1954 and could find no positive correlation between the increased height of the T waves and the serum potassium levels determined immediately after the race. However, two subjects were studied 8, 21 and 30 hours after the race where it was found that the T waves were lowered as serum potassium rose. Seldon[15] in 1958 attributed T wave changes to carbohydrate ingestion, fear and sympathetic tone. Gardberg and Rosen,[14] however, believed T wave variation to be caused by postural change in the heart's electrical position.

This experiment was not in the first instance designed to define the causes of any observed changes but to document these findings. It is suggested that the changes reported are probably physiological under the condition of maximal effort in healthy, young, trained individuals.

The resting graphs and recovery graphs of all subjects are on file in the main library of the University of British Columbia. All procedures were supervised by C. G. Campbell, M.D., M.Sc., F.R.C.P.[C], member of the attending staff at Vancouver General Hospital's Heart Station and clinical instructor at the University of British Columbia. Analysis of the ST segment was supervised by J. A. Osborne, M.D., F.R.C.P.[C], Assistant Director of Cardiology at Vancouver General Hospital's Heart Station.

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